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Echocardiographic assessment of abnormal left ventricular relaxation in man[1]

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In 64 patients requiring cardiac catheterization for chest pain, echocardiograms showing anterior mitral leaflet and left ventricular cavity simultaneously were recorded. These were digitized and their first derivatives computed in order to study time relations between mitral valve and left ventricular wall movement in early diastole. In 10 patients with normal left ventricular angiograms and coronary arteriograms, mitral valve opening began 1·1 ± 9·3 ms (mean ± SD) before the onset of outward wall movement, and reached peak opening velocity 2·0 ± 13 ms after maximum rate of change of dimension. Virtually identical time relations were seen in 15 patients with normal left ventricular angiograms but with obstructive coronary artery disease (3·6 ± 9·3 ms and 0·7 ± 7·3 ms, respectively). These close relations were lost in patients with segmental abnormalities of contraction on left ventricular angiogram. In 19 such patients with normal septal motion, outward wall movement began 53 ± 31 ms before the onset of anterior movement of the mitral valve leaflet, and this isovolumic wall movement accounted for 31 per cent of the total diastolic excursion. In 9 patients with reversed septal movement, these abnormalities were greater, 92 ± 39 ms and 33 per cent, respectively, while in 11 patients with diffuse left ventricular involvement they were small, 5·5 ± 13 ms and 3 per cent. Frame-by-frame digitization of cineangiograms was used to confirm these findings which appear to reflect an abnormal change in left ventricular cavity shape during isovolumic relaxation.

Although systolic abnormalities of left ventricular contraction have been well documented in ischaemic heart disease (Herman and Gorlin, 1969; Baxley and Reeves, 1971), only recently have diastolic abnormalities of left ventricular relaxation been noted. Within the past 4 years, several angiographic studies have described the onset of outward wall movement occurring before mitral valve opening (Ruttley *et al.*, 1974; Altieri, Wilt, and Leighton, 1973; Hamby *et al.*, 1974; Wilson *et al.*, 1975), and the term segmental early relaxation phenomenon (SERP) has been used to describe this event. The diagnostic significance of these movements, however, is not clear since they were found whether or not ischaemic heart disease was present. The purpose of the present study was to see whether SERP could be detected by echocardiography in patients undergoing cardiac catheterization for the evaluation of chest pain, and to determine its relation, when present, to angiographic findings. In order to do this, echocardiograms of the anterior mitral valve leaflet and left ventricular cavity were recorded simultaneously at 100 mm/s, and a simple digitizing technique was used to compute instantaneous mitral valve velocity, left ventricular dimension, and its rate of change, so that the relation between mitral valve and wall movement in early diastole could be determined.

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Subjects and methods

Subjects

Echocardiographic studies were performed on 64 patients who underwent cardiac catheterization for the evaluation of chest pain. Their ages ranged from 32 to 61, and 9 were women. All were in sinus rhythm and were studied within 24 hours of catheterization. They were selected only in so far as they were patients in whom it was possible to obtain acceptable echocardiographic records.

They were divided according to the findings at

catheterization into the following groups:

Group 1 Ten patients with chest pain, but without occlusive coronary artery disease. Left ventricular haemodynamics and angiograms were also normal, and no cardiac cause could be found for their symptoms.

Group 2 Fifteen patients with significant occlusive disease (greater than 70%) of one or more coronary artery, but with normal left ventricular haemodynamics and angiograms. None of these patients had electrocardiographic evidence of a previous transmural infarction.

Group 3 Twenty-eight patients with significant occlusive coronary disease and segmental contraction abnormalities on angiogram; 20 of these had electrocardiographic evidence of a previous transmural myocardial infarction, and 14 had a raised left ventricular end-diastolic pressure (greater than 13 mmHg (1·7 kPa)). They were further divided according to septal movement at the time of echocardiographic study, into 2 subgroups: (3a) 19 with normal septal movement and (3b) 9 with reversed septal movement (anterior movement during systole).

Group 4 Eleven patients with generalized impairment of left ventricular contraction and diminished ejection fraction (less than 30%). All had raised left ventricular end-diastolic pressure. Significant occlusive coronary artery disease was found in 4 of these patients who underwent coronary angiography, and a presumptive diagnosis of ischaemic cardiomyopathy was made in the rest from their histories.

Echocardiographic methods

An Ekoline 20 ultrasonoscope (frequency 2·25 MHz; repetition frequency 1000/s) was used to make all measurements, and the output was displayed on a Cambridge multichannel photographic strip-recorder at a rate of 100 mm/s with a simultaneous electrocardiogram. The patients were studied in the left lateral oblique position with the transducer in the 3rd or 4th left intercostal space. The aortic root was first identified, and then the transducer was angled down through the mitral ring until echoes were obtained simultaneously from the anterior mitral valve leaflet, interventricular septum, and endocardium of the posterior wall (Fig. 1). Measurements were only made on those recordings showing clear, continuous echoes throughout a complete cardiac cycle. In addition, the posterior leaflet had to be recorded during at least part of diastole before the record was accepted. Wall movement was thus studied only at the level of the mitral valve.

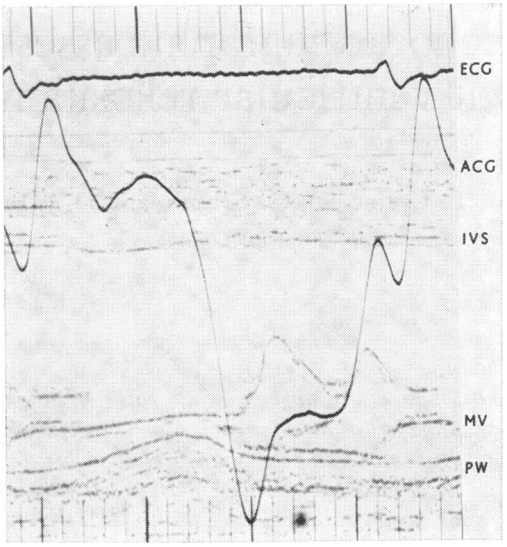

FIG. 1 *Echocardiogram of the left ventricular cavity from a patient with an abnormal left ventriculogram, showing interventricular septum (IVS), mitral valve cusps (MV), and posterior wall (PW). An apex cardiogram (AGG) is superimposed.*

Angiographic methods

Left heart catheterization was performed retrogradely via the femoral artery. Immediately after left ventricular pressures were measured in the routine manner with a fluid-filled catheter, ventriculograms were obtained with the patient in the posteroanterior position. From 30 to 50 ml Triosil 75 per cent was injected into the left ventricle at a rate of 25 ml/s, and cine film was exposed at approximately 50 frames/s. An electrocardiogram was recorded throughout the injection and superimposed on the cine frames so that the film speed could be measured exactly. Calibration was carried out by means of a grid exposed at midchest level. Coronary arteriograms were performed using 3 to 8 ml injections of Urografin 76 by the Judkins technique and recorded in multiple views.

Digitization

The method used to digitize the echocardiograms has been described elsewhere in detail (Gibson and Brown, 1973). The echoes of the septum, posterior wall, and anterior mitral leaflet were traced with a DMAC digitizing table interfaced with an IBM

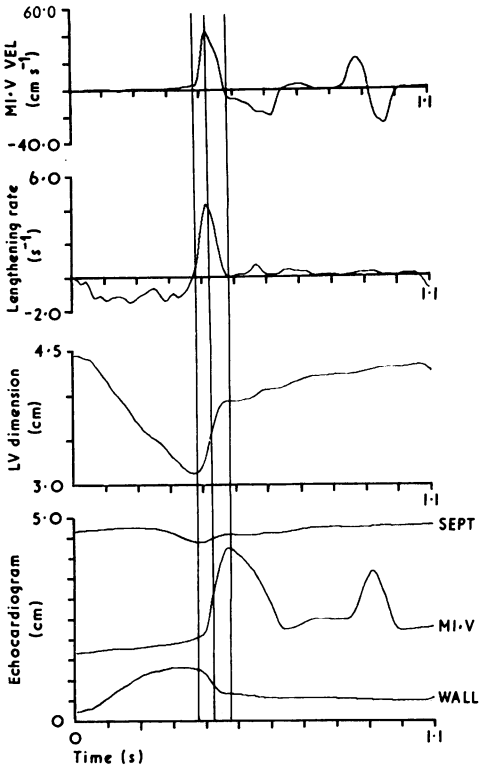

FIG. 2 *Digitized echocardiogram from a patient with a normal left ventriculogram. The lowest panel represents septal, mitral valve, and posterior wall echoes. Above are plotted left ventricular dimension (D), normalized lengthening rate (dD/dt/D), and anterior mitral valve cusp velocity. The three vertical lines correspond to the onset, peak, and discontinuity in outward wall movement.*

1800 computer. Strings of co-ordinates were generated for approximately 100 points on each of these echoes throughout a complete cardiac cycle, together with calibration signals representing 0·5 s, 1 cm, and the RR interval of the beat being studied.

Left ventriculograms of a representative number of patients from each group were also digitized in a similar manner (Gibson and Brown, 1975a). Ectopic and postectopic beats were excluded. Successive frames of the beat to be studied were projected on to the digitizing table, and the opacified ventricular cavity outlines were traced with the cursor. Strings of co-ordinates were generated for approximately 50 points on each frame, together with a reference point on the border of the cine frame.

Analysis of data

The echocardiograms were analysed as previously described (Upton, Gibson, and Brown, 1976). From the stored digitized data, plots were made of the positions of wall and anterior mitral leaflet, instantaneous leaflet velocity, left ventricular dimension (D), and normalized rate of change of dimension (dD/dt/D) (Fig. 2). Peak velocities of leaflet movement during early diastolic opening (D-E) and mid-diastolic closure (E-F) were measured directly from the plots, as were the end-diastolic dimension and peak rate of wall movement during diastole. In addition, the time relation between wall and mitral leaflet movement in early diastole was analysed by measurement of the intervals between the time of (1) the onset of outward wall movement and that of mitral leaflet opening movement, (2) the peak rate of change of dimension and peak opening velocity of the leaflet, and (3) a discontinuity in the rate of wall movement previously

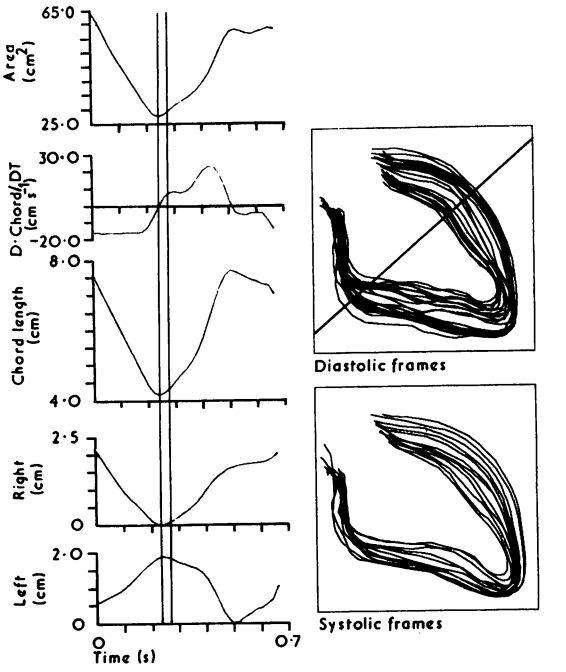

FIG. 3 *Digitized left ventriculogram, showing systolic (below) and diastolic (above) frames superimposed. The diagonal line represents the position of the chord studied. On the left are shown, from below, movement of the right and left sides of the cavity plotted against time, chord length and its rate of change, and cavity area. Vertical lines represent the timing of minimum cavity area and mitral valve opening.*

TABLE 1 *Echocardiographic data (mean values ±1 standard deviation)*

	Heart rate (beats/min)	*Peak opening (D-E) velocity (mm/s)*	*Peak closing (E-F) velocity (mm/s)*	*Peak dD/dt/D (s^{-1})*	*End-systolic dimension (cm)*
Chest pain with normal coronaries (10)	67 ±9	400 ±60	240 ±60	3·1 ±0·48	3·2 ±0·26
IHD with normal LV angiograms (15)	69 ±7	360 ±80	210 ±50	2·9 ±0·60	3·4 ±0·38
IHD with abnormal LV angiograms (19)	73 ±11	430 ±90	210 ±60	2·2 ±0·63	4·1 ±0·72
IHD with abnormal LV angiograms and RSM (9)	70 ±8	420 ±120	230 ±70	1·7 ±0·53*	4·1 ±0·67
Cardiomyopathies (11)	87 ±14***	460 ±70*	210 ±40	1·4 ±0·40**	6·3 ±0·79***

*P<0·05, **P<0·01, ***P<0·001 } Statistical significance of difference from group with normal coronaries.
IHD, ischaemic heart disease; LV, left ventricular; RSM, reversed septal movement.

TABLE 2 *Time interval from left ventricular wall to mitral valve movement (ms) (mean values ±1 standard deviation)*

	Onset	*Peak*	*Discontinuity*
Chest pain with normal coronary arteries (10)	−1·1 ±9·3	2·0 ±13	−1·3 ±20
IHD with normal LV angiograms (15)	3·6 ±9·3	0·7 ±7·3	−2·3 ±16
IHD with segmental contraction abnormalities on angiogram (19)	53 ±31**	37 ±28**	33 ±26**
IHD with segmental contraction abnormalities on angiogram and RSM on echocardiogram (9)	92 ±39**	23 ±23*	35 ±24**
Cardiomyopathies (11)	5·5 ±13	7·3 ±18	−3·3 ±13

*P<0·01, **P<0·001 } Statistical significance of difference with respect to group with normal coronary arteries.

shown to coincide with the end of rapid filling (Prewitt *et al.*, 1975) and a corresponding discontinuity in the leaflet velocity tracing during mid-diastolic closure. In those patients in whom outward wall movement occurred before the onset of mitral leaflet opening movement, the increase in left ventricular dimension which occurred over this interval was measured and expressed as a percentage of total dimensional change during diastole. This technique also made it possible to delineate the different patterns of left ventricular wall movement which occurred during isovolumic relaxation.

The left ventriculograms and coronary angiograms were analysed and reported upon by two independent observers. In addition, in representative patients from each group, cavity outlines were plotted superimposed on one another from the stored data (Fig. 3). Those frames from the start of the beat being studied to the one with the smallest area were taken as systole and the remainder as diastole. Cavity area was derived by numerical integration and also plotted. In addition, a transverse diameter was drawn across the cavity outlines, displayed on a Ferranti MD4 visual display unit with the position of the cursor manually adjusted to represent the path of the ultrasound beam. Plots were thus made of the 'right' and 'left' sides of each chord analogous with the posterior wall and septal echoes, and left ventricular dimension and its rate of change were derived at this level. The time of mitral valve opening was determined by frame-by-frame analysis of the left ventriculograms and identified as the point when the first unopacified blood was seen in the left ventricle. The interval between the onset of outward wall movement and mitral valve opening and the percentage of total diastolic change in transverse dimension and cavity area during this interval could thus be assessed by a second method.

Results

Echocardiography (Tables 1 and 2)

Group 1 In this group, with chest pain and normal coronary arteries, the heart rate was 67 ±9, the peak lengthening rate was 3·1 ±0·48 s^{-1}, and the end-systolic dimension was 3·2 ±0·26 cm (Table 1). These values are similar to those previously reported for normal subjects (Gibson and Brown, 1973). The peak opening velocity was 400 ±60 mm/s,

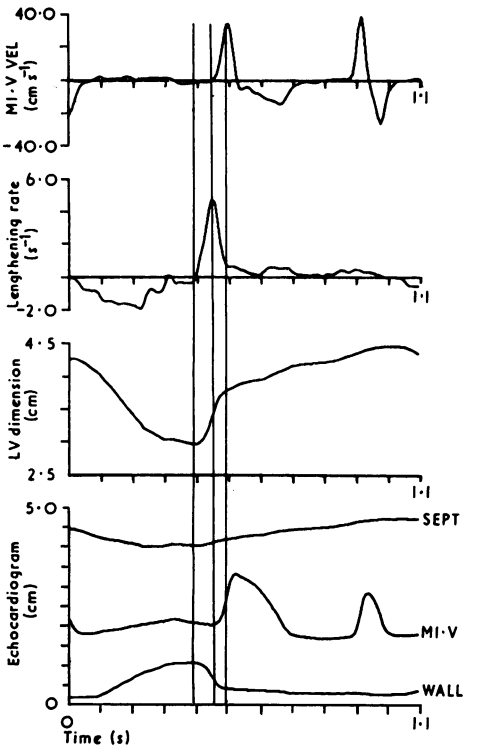

FIG. 4 *Digitized echocardiogram from a patient with an abnormal left ventriculogram. The layout is the same as in Fig. 2. The onset of mitral valve opening is delayed with respect to that of the increase of dimension, but coincides with its peak rate of change.*

and the peak mid-diastolic closure rate was 240 ± 60 mm/s. Again, these values agree with those previously reported in normal subjects (Upton *et al.*, 1976). In early diastole, there was a close time relation between left ventricular wall and mitral valve movement. The onset of mitral valve opening movement began 1·1 ±9·3 ms before the onset of outward wall movement (Table 2). The peak opening velocity of the valve followed the peak rate of wall movement by 2·0 ±13 ms, and later a discontinuity in the velocity tracing during mid-diastolic closure began 1·3 ±20 ms before a similar discontinuity in wall movement (Fig. 2). These corresponding events in mitral valve and wall movement occurred simultaneously, since the differences are not significantly different from zero. This close time relation has previously been noted in normal subjects (Upton *et al.*, 1976).

Group 2 Using the same indices as above, this group with ischaemic heart disease and normal left ventriculograms could not be distinguished from group 1.

Group 3a These patients, with ischaemic heart disease and segmental contraction abnormalities on their left ventriculograms, could not be distinguished from group 1 with respect to heart rate, peak mitral valve opening and closing velocities, peak lengthening rate, and end-systolic dimension. The onset of mitral valve opening movement, however, began 53 ±31 ms after the onset of outward wall movement, and during this isovolumic interval, 31 per cent of total diastolic increase in transverse dimension occurred. The peak and discontinuity of the mitral valve velocity tracing were also delayed with respect to the corresponding events in wall movement with the time intervals being significantly increased compared with group 1 ($P < 0.001$) (Fig. 4).

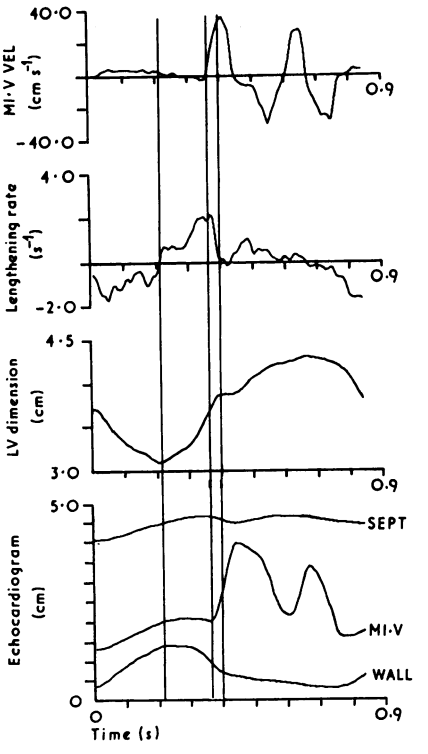

FIG. 5 *Digitized echocardiogram from a patient with an abnormal left ventricular angiogram. The layout is the same as for Fig. 2. Septal movement is reversed. The onset of mitral valve opening corresponds with peak rate of increase of dimension rather than with its onset.*

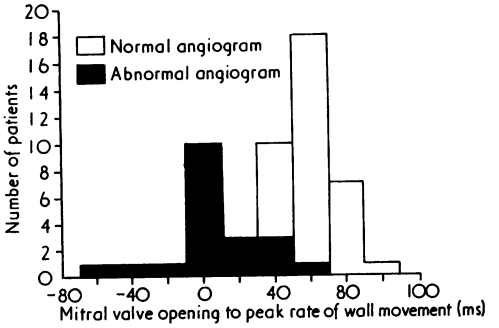

FIG. 6 *Distribution within the patient population of the time interval between the onset of mitral valve opening and the peak rate of increase in dimension in patients with normal and abnormal left ventriculograms.*

Group 3b In this group, with segmental contraction abnormalities on left ventriculogram and reversed septal movement on echocardiogram, mitral valve opening movement began 92 ± 39 ms after the onset of outward wall movement, significantly longer than in group 3a ($P < 0{\cdot}01$), with 33 per cent of total diastolic increase in transverse dimension occurring during this interval. Again, the peak and discontinuity of the mitral valve velocity tracing were also significantly delayed with respect to the corresponding events in wall movement. In addition, the peak lengthening rate of $1{\cdot}7 \pm 0{\cdot}53$ s^{-1} was significantly less than in group 1 (Fig. 5).

The relation between the onset of mitral valve opening and the peak rate of wall movement in patients with normal and abnormal left ventriculograms is shown in Fig. 6. In those with normal angiocardiograms, mitral valve opening occurred most frequently 60 ms before the time of peak rate of change of dimension; in the majority of patients with segmental abnormalities of contraction, the two events were simultaneous. The scatter about these mean values was similar in the two groups.

Group 4 This group, with generalized impairment of left ventricular contraction and decreased ejection fraction on angiogram (cardiomyopathies), could easily be distinguished from group 1 with the usual criteria. The resting heart rate was significantly higher at 87 ± 14 beats/min ($P < 0{\cdot}001$), the end-systolic dimension was raised significantly at $6{\cdot}3 \pm 0{\cdot}79$ cm ($P < 0{\cdot}001$), and the peak lengthening rate was reduced at $1{\cdot}4 \pm 0{\cdot}40$ s^{-1} ($P < 0{\cdot}01$). In this group, however, the onset of mitral valve opening was only $5{\cdot}5 \pm 13$ ms after onset of outward wall movement, and only 3 per cent of the total diastolic excursion occurred in this interval. These values were not significantly different from those in group 1. In addition, the peak and the discontinuity of the mitral valve velocity tracing corresponded closely to the corresponding peak and discontinuity in wall movement, the time differences between the two not being significantly different from zero.

Angiography (Table 3)

Group 1 In 5 representative patients from this group, the onset of mitral valve opening occurred 85 ± 13 ms after the onset of outward wall movement, and 21 per cent of the total increase in transverse dimension occurred in this time interval. This was associated with a $15 \pm 3{\cdot}9$ per cent increase in cavity volume from the end-systolic value (Fig. 3).

Group 2 The results in 5 patients in this group studied angiographically were not significantly different from those in group 1.

Group 3 In 10 representative patients from this group with segmental abnormalities shown by left ventriculography, mitral valve opening occurred 140 ± 36 ms after the onset of outward wall movement. Though 41 per cent of total diastolic movement along the transverse diameter occurred during this interval, there was only a 12 per cent increase

TABLE 3 *Angiographic correlation with echocardiographic findings (mean ±1 standard deviation)*

	Time interval from onset of outward wall movement to MV opening (ms)	*Per cent total wall movement before MV opening*	*Per cent total volume increase before MV opening*
Chest pain with normal coronary arteries (5)	85 ± 14	$21 \pm 4{\cdot}0$	$15 \pm 3{\cdot}9$
IHD with normal LV angiograms (5)	74 ± 24	$16 \pm 9{\cdot}1$	$14 \pm 7{\cdot}7$
IHD with segmental contraction abnormalities on LV angiogram (10)	140 ± 36**	41 ± 16**	$12 \pm 8{\cdot}1$
Cardiomyopathies (5)	65 ± 22	$17 \pm 6{\cdot}3$	$11 \pm 8{\cdot}5$

**$P < 0{\cdot}001$ Statistical significance of difference from group with normal coronaries.
MV, mitral valve.

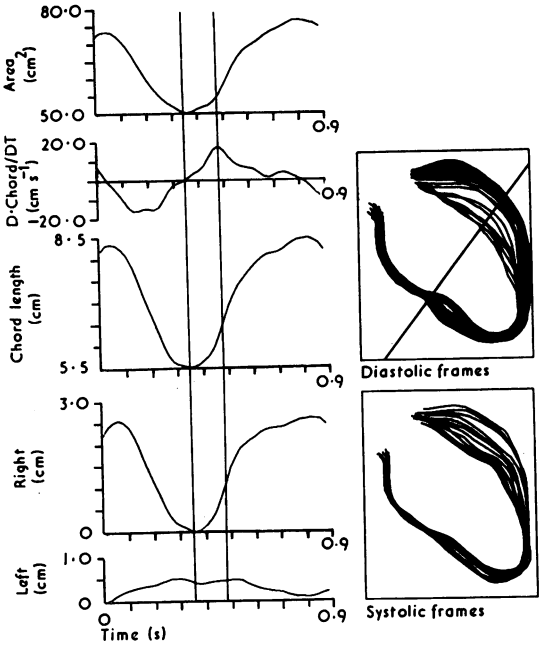

FIG. 7 *Digitized left ventricular angiogram from a patient with inferior hypokinesia. Layout as in Fig. 3. A significant increase in chord length has occurred before mitral valve opening.*

in area, the latter value not being significantly different from normal (Fig. 7).

Group 4 Results in 5 patients in this group studied angiographically were not significantly different from those in group 1, with respect to the interval between the onset of outward wall movement and mitral valve opening and the percentage of transverse diameter and cavity area increase before mitral valve opening.

Discussion

The present study has shown considerable variation in early diastolic events in patients presenting with anginal pain. In those with normal left ventriculograms, with or without coronary artery disease, the pattern of left ventricular wall movement was normal in early diastole, in that the onset and peak rates of outward wall movement were synchronous with those of anterior mitral valve cusp movement. In patients with angiographically demonstrable segmental abnormalities of contraction, however, the normal time relation between left ventricular wall movement and that of the mitral valve was disturbed. In almost all of them, outward left ventricular wall movement started before mitral valve opening, suggesting that it was occurring in the period of isovolumic relaxation, and thus was associated with a change in cavity shape rather than with filling. Isovolumic left ventricular shape changes have previously been studied angiographically, and have been described in normal subjects, and in patients with prolapsing mitral valve cusp (Gooch *et al.*, 1972) or ischaemic heart disease. They have previously not been detected by echocardiography, though an abnormality of systolic mitral motion described by Greenwald *et al.* (1975) may be explicable on the basis of mitral valve opening being late when compared with the onset of posterior movement of the mitral ring or posterior left ventricular wall.

In spite of its obvious limitations in depicting only regional function, echocardiography has certain advantages for studying left ventricular wall movement during early diastole. It enables the endocardium to be recognized unequivocally throughout this period of the cardiac cycle, when angiographic methods are open to question. It also allows mitral valve cusp movement to be observed and compared with that of the ventricular wall, and it avoids the introduction of a catheter into the left ventricle, so eliminating the possibility that early diastolic wall movement is caused by artefactual valvular regurtitation. Using echocardiography, it was possible to show that left ventricular wall movement was very abnormal in patients with segmental abnormalities of contraction shown by angiography. Instead of the normal synchronous movement of left ventricular wall and mitral valve cusp, there was a very significant increase in transverse dimension before the onset of ventricular filling. This resulted in the time of mitral valve opening being considerably delayed with respect to the time of minimum dimension. The commonest pattern of wall movement was of increasing rate of change of dimension, reaching a peak at the time of onset of forward movement of the anterior cusp. After this, the rate of increase of dimension dropped abruptly with a corresponding discontinuity on the dimension trace itself. Less commonly, the initial rate of outward movement was slow until the onset of forward movement of the anterior cusp but increased to normal values thereafter. This pattern merged into the normal one when the onset of mitral valve and wall movement were simultaneous. In 3 patients wall movement was very abnormal, with peak rates being registered before mitral valve opening, so that the rate of increase of dimension was already low at the start of filling, and declined still further until the succeeding left atrial systole. However, when considered together, all patients

with segmental systolic abnormalities behaved as a single group, values for the interval between the onset of mitral valve and dimension change showing a normal frequency distribution about a value in which peak rate of change of dimension coincided with the onset of forward movement of the anterior cusp.

A surprising feature was the frequency with which these changes were detected by a method capable of studying only a small part of the ventricle. This suggests that the abnormality of early relaxation may be a generalized one, though related to a primary local disturbance of systolic function, possibly dependent on regional ischaemia. The direction and magnitude of the changes in dimension were unrelated to the localization of the systolic abnormality, but the relative delay in the onset of anterior movement of the mitral valve cusp was significantly greater when there was reversed septal movement. In contrast to patients with segmental abnormalities, these changes were not present when contraction was uniformly impaired because of severe ischaemic heart disease, stressing the importance of regional involvement in their genesis.

The relation between left ventricular dimension changes and mitral valve opening studied by angiography was significantly different in patients with normal left ventriculograms from that shown by echocardiography. Angiography showed that mitral valve opening was not synchronous but delayed with respect to the onset of outward wall movement, by a mean value of 85 ms, and during this period a mean increase of 21 per cent of the total diastolic excursion of dimension had occurred. The reason for this discrepancy is not clear, but it is likely to be the result of different methods of timing mitral valve opening. This was defined by echocardiography as the start of forward movement of the anterior cusp, and by angiography as the time of first appearance of unopacified blood within the left ventricular cavity. In some angiograms mitral valve movement itself was apparent as an early inward movement of the boundary of the cavity, followed by mid-diastolic closure, and in these records the frame in which unopacified blood was first considered to be present within the cavity coincided with peak opening rather than with the onset of forward movement (unpublished observations). This suggests that the angiographic definition of the onset of normal left ventricular filling identifies a point in time at least 50 ms later than that defined by the echocardiographic method. This result is compatible with that of animal studies (Pohost *et al.*, 1975) which have shown that the onset of forward movement of the anterior cusp precedes the start of left ventricular filling, taken as the time of crossover between left atrial and left ventricular pressures.

In patients with segmental contraction abnormalities, however, echocardiography was in agreement with angiography in showing a significant delay in mitral valve opening, both with respect to minimum dimension and cavity area, and during this period the increase in transverse dimension was significantly greater than that occurring in patients with normal left ventriculograms. This abnormality was present regardless of the site of the disturbance of contraction, being seen whether or not the dimension studied passed through the affected area. Related localized abnormalities have previously been described using angiography in patients with ischaemic heart disease, but these have been defined in terms of regional wall movement rather than as dimension changes as in the present study. During isovolumic relaxation, both inward and outward wall movement may occur in different parts of the cavity, the former being closely related to the pattern of coronary artery involvement, and the latter being, in part, compensatory. This reflects the fact that aortic and mitral valves are closed so that left ventricular volume cannot change. The resulting change in left ventricular cavity shape before mitral valve opening is always towards a more circular configuration in a single projection (Gibson, Prewitt, and Brown, 1976) and it is this that appears to be the basis of the dimension changes observed in the present study. An increase in minor diameter is a necessary consequence of cavity shape becoming more spherical, thus explaining how a regional disturbance anywhere in the ventricle can give rise to this single abnormality, detectable by echocardiography or angiography. Both methods show the frequent occurrence of increasing velocity of outward wall movement until mitral valve opening, suggesting that the isovolumic state is a necessary condition for the maintenance and propagation of this abnormality of wall movement, which resolves once ventricular filling begins. Patients with dilated left ventricular cavities and low ejection fraction show no significant shape change between systole and diastole (Gibson and Brown, 1975b) and so would not be expected to show any significant change in cavity configuration during isovolumic relaxation.

The present results clearly show that the echocardiographic dimension cannot be used to assess left ventricular filling in patients with systolic abnormalities of contraction resulting from ischaemic heart disease. This is particularly the case for estimates of peak rates of wall movement, which in the majority of cases, either preceded or were synchronous with the onset of mitral valve movement, and so could not possibly have been related

to the rapid phase of ventricular filling. Similarly, the discontinuity on the dimension trace, which in normal subjects seems to indicate the end of rapid filling, in those with segmental abnormalities usually represented its start. It was not possible, either, to deduce the presence of these disturbances of left ventricular function from observations of the movement of the anterior cusp of the mitral valve during its opening or mid-diastolic closure. Whatever the mechanism of a reduced diastolic closure rate in left ventricular disease, it is not the result of segmental or generalized abnormalities of left ventricular contraction, a raised end-diastolic pressure, or incoordinate wall movement during filling. However, the results indicate that the versatility of the echocardiographic method can be increased in such patients by simultaneously recording echoes from the mitral valve as well as the left ventricular cavity and by using simple digitizing techniques. This allows isovolumic wall movements to be detected and distinguished from those caused by ventricular filling, and suggests that, when such disturbances are present, there is a high probability of the left ventricular angiogram showing segmental abnormalities of function.

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